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Transient epileptic amnesia versus transient global amnesia: aspects of differential diagnosis

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SUMMARY

Transient global amnesia (TGA) and transient epileptic amnesia (TEA) are rare phenomena in clinical practice that manifest as transient cognitive amnestic impairments. Despite the similarity in clinical picture, such conditions are pathogenetically heterogeneous and require different therapeutic approaches. TGA is a clinical syndrome characterized by sudden anterograde amnesia of the event lasting up to 24 hours, lacking focal neurological symptoms, and not prone to recurrence. Mimicking TGA, TEA often occurs manifested as epileptic seizures with impaired awareness of varying duration, including long-term (more than 24 hours), as a variant of focal epilepsy. TEA is characterized by recurrent episodes, combination with other manifestations of epilepsy, and comorbidity with neurodegenerative diseases (dementia). For differential diagnosis, it is necessary to use prolonged video-electroencephalographic monitoring with sleep recording, neuroimaging methods (brain magnetic resonance imaging, positron emission tomography), psychological testing, biochemical examination for markers of neurodegeneration.

KEYWORDS

Epilepsy, transient global amnesia, TGA, transient epileptic amnesia, TEA, cognitive impairment, dementia.

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Транзиторная эпилептическая амнезия и транзиторная глобальная амнезия: аспекты дифференциальной диагностики

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РЕЗЮМЕ

Транзиторная глобальная амнезия (ТГА) и транзиторная эпилептическая амнезия (ТЭА) – редко встречающиеся в клинической практике феномены, которые проявляются преходящими когнитивными амнестическими нарушениями. Несмотря на схожесть клинической картины, эти состояния патогенетически разнородны и требуют различных терапевтических подходов. ТГА – клинический синдром, характеризующийся внезапной антероградной амнезией события

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длительностью до 24 ч без очаговых неврологических симптомов, не склонный к рецидивированию. Под маской ТГА часто протекает ТЭА – эпилептические приступы с нарушением осознанности различной длительности, включая долгосрочные (более 24 ч), как вариант фокальной эпилепсии. Для ТЭА характерны повторяемость эпизодов, сочетание с другими проявлениями эпилепсии, коморбидность с нейродегенеративными заболеваниями (деменцией). Для дифференциальной диагностики необходимо использовать пролонгированный видеоэлектроэнцефалографический мониторинг с записью сна, методы нейровизуализации (магнитно-резонансную томографию, позитронно-эмиссионную томографию головного мозга), психологическое тестирование, биохимическое исследование маркеров нейродегенерации.

КЛЮЧЕВЫЕ СЛОВА

Эпилепсия, транзиторная глобальная амнезия, ТГА, транзиторная эпилептическая амнезия, ТЭА, когнитивные нарушения, деменция.

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INTRODUCTION / ВВЕДЕНИЕ

The phenomenon of transient global amnesia (TGA) is well known to clinicians and is described in detail. It is characterized by sudden anterograde amnesia usually lasting from 4 to 6 hours, less often up to 24 hours, with other neurological functions usually being preserved [1–3].

However, under the guise of TGA, transient epileptic amnesia (TEA) often occurs including epileptic seizures with impaired awareness of various durations, including long-term, so-called twilight consciousness disorders or trances, which are essentially a manifestation of focal temporal limbic epilepsy that occurs later in life.

The similarity of the clinical symptoms between TGA and TEA causes marked difficulties in the differential diagnosis of these pathogenetically heterogeneous conditions, given their episodicity, high comorbidity with cerebrovascular and neurodegenerative diseases, as well as the relationship with physical and emotional excesses.

TRANSIENT GLOBAL AMNESIA/ ТРАНЗИТОРНАЯ ГЛОБАЛЬНАЯ АМНЕЗИЯ

The prevalence of TGA comprises 3.4–10.4 cases per 100 thousand population per year, reaching 23.5 cases per 100 thousand population per year in patients over 50 years of age [4], usually lacking gender bias in TGA incidence, but there are separate reports describing a higher prevalence in middle-aged and elderly women, with female-to-male ratio reaching 4:1 [5].

Most often, TGA is a single event that rarely recurs: the relapse rate with an average follow-up of at least 3 years ranges from 2.9% to 23.8%. The wide relapse range probably reflects different criteria for determining TGA and the relatively low number of patients with recurrent episodes [6, 7].

TGA attacks usually have a specific trigger. The TGA development can be preceded by physical exertion, emotional stress, exposure to an excessively prominent temperature factor, pain, physical overexertion, and a Valsalva test [8]. It is suggested that TGA can be caused by transient intracranial venous hypertension [1], transient ischemic brain damage (in the basin of the posterior cerebral artery), hypertensive crisis, and ischemic cerebral stroke [9, 10]. The risk group includes patients after coronary angiography. The relationship between TGA and migraine is described, which does not exclude common genetic or pathophysiological mechanisms underlying such diseases [11–13].

TGA is verified based on a clinical diagnosis using the following criteria [4, 5]:

– verified anterograde amnesia, confirmed by information from a reliable witness of the attack;

– the patient has an unaltered level of consciousness or orientation in own personality;

– cognitive deficits characterized solely by amnesia of the TGA event;

– absence of focal neurological symptoms (aphasia, apraxia, homonymous hemianopsia, etc.) or convulsive seizures and their consequences (Todd's paralysis, etc.);

– no history of traumatic brain injury or epilepsy (taking anticonvulsants or epileptic seizures in the last 2 years);

– symptoms regressed within 24 hours;

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– mild vegetative symptoms, headache, nausea and/or dizziness may occur within 1 day (acute phase of TGA).

Electroencephalography (EEG) in TGA patients, as a rule, reveal no specific pathological patterns that helps to exclude epileptic seizures, postictal state, and non-convulsive epileptic status [2, 14, 15].

Approximately 12% of patients demonstrate different variants of normal EEG (rhythmic or wave activity), which are mistakenly interpreted by neurologists as epileptiform discharges in about 30% of cases [16]. Among such EEG phenomena, subclinical rhythmic electroencephalographic discharges in adults (SREDA) is a very rare (prevalence 0.04–0.07%) benign variant that mimics ictal epileptiform discharges and can lead to an erroneous diagnosis of epilepsy. SREDA consists of a 2–6 Hz delta-theta rhythm (usually a 5–6 Hz theta rhythm) with a spike morphology, generalized distribution (peaking in the posterior regions) usually manifested in the elderly (average age 52 years) during wakefulness or in a state of superficial sleep (stage 1 NREM sleep[1]), as well as during hyperventilation lasting 40–80 seconds, with a sudden onset and end able to mimic epileptic seizures, thereby complicating diagnostics. The pathophysiology of this phenomenon is unknown, but it is assumed that it may be caused by local cerebral hypoxia at the border of the parietal-occipital-temporal-parietal regions. Atypical variants may also occur (e.g., SREDA of the frontal region), while slowing down in the posterior parts or suppressing the background alpha rhythm is not observed [5, 17, 18].

Neuroimaging studies, mainly brain magnetic resonance imaging (MRI) (T2-diffusion sequences), can verify the etiology and topography of brain changes in TGA patients. Diffusion MRI can confirm TGA diagnosis in 70.6% of patients (69.1% with low diagnostic reliability in the absence of a witness to the event, and 93% – with high diagnostic accuracy). if there is a reliable TGA witness), if it is performed within the first 20 hours after the onset of the clinical manifestation of the event [19]. EEG detection of the SREDA phenomenon in TGA patients can play a key diagnostic role in MRI-negative patients (lacking pathological brain changes).

TGA differential diagnosis should be made with acute cerebral circulatory disorders (transient ischemic attack or stroke in the posterior cerebral artery basin), impaired glucose tolerance (hypoglycemia), toxic-metabolic encephalopathy due to intoxication with psychoactive substances (alcohol, sympathomimetics (cocaine, etc.)), undesirable side effects of pharmacotherapy with benzodiazepines, tricyclic antidepressants, neuroleptics, etc., TEA, psychogenic amnesia (a violation of personal identity in conversion disorders), post-traumatic retrograde amnesia, Wernicke–Korsakov syndrome, cognitive disorders in neuroinfections (herpetic encephalitis, etc.) and autoimmune limbic encephalitis.

TGA prognosis is favorable and requires no specific treatment. The relapse rate is 2.9–23.8%, and a higher risk of TGA recurrent episodes is described in patients with migraines. Despite the benign course of this disease, TGA episodes can cause anxiety in both patients and family members. TGA can mimic or mask various neuropsychiatric diseases, which has negative consequences for certain professions [20–23].

TRANSIENT EPILEPTIC AMNESIA / ТРАНЗИТОРНАЯ ЭПИЛЕПТИЧЕСКАЯ АМНЕЗИЯ

TEA is a manifestation of late-onset epilepsy, which is characterized by attacks of transient amnesia and inter-onset cognitive impairment, which can be a sign of neurodegenerative diseases such as dementia.

Perhaps, J. Hughlings-Jackson is the first author who suggested in 1888 that transient amnesia may be the sole or most prominent manifestation of an epileptic seizure, calling it an “intellectual aura” while describing his patient's illness [24]. The term “transient epileptic amnesia” was introduced in 1990 to emphasize the existence of a special form of epilepsy that causes transient amnesic seizures superficially resembling TGA attacks [25–27]. Subsequent descriptions emphasize the association between TEA with “atypical” forms of memory impairment, including rapid deterioration of long-term memory, and the disproportionality of autobiographical and topographical amnesia [28].

EEG is the most important TEA diagnostic tool. Epileptiform changes in TEA are detected in 35% of cases: interictal changes involving one-or two-sided acute waves (1/3 of cases), non-specific focal slow waves (1/3), diffuse low-amplitude rapid activity in the temporal region with a repetitive rhythm extending from one to another hemisphere. Epileptiform patterns on the EEG are recorded in the frontotemporal regions and may show a bilateral pattern. While performing long-term video-EEG monitoring with sleep recording, the detectability of epileptiform activity increases up to 50% [12]. It is proved that sleep is important for memory consolidation, and pathological neuronal activity during sleep and nocturnal subclinical seizures are the pathogenetic basis of mnestic disorders both in TEA and in the interictal period. In addition, concomitant sleep disorders, such as obstructive sleep apnea, can increase seizure frequency.

Thus, long-term EEG recording, including sufficient sleep recording, can be crucial both for diagnosing TE, especially when short-term EEG studies are uninformative, and for verifying uncontrolled subclinical seizures with cognitive and behavioral disorders in case of ineffective prescribed antiepileptic therapy [29]. The complexity of making a correct diagnosis and the late onset of antiepileptic drug therapy (AEDs) elevate the risks of deteriorated patients’ quality of life and health care costs, so for patients with amnestic disorders and a normal routine EEG picture it is important to perform long-term video-EEG monitoring as early as possible.

[1] NREM sleep – non-rapid eye movement sleep.

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The neuroimaging data in TEA are usually uninformative, and clinically significant abnormalities in brain MRI were detected only in 5% of cases. However, studies involving MRI post-processing, automated MR-volumetry followed by a "manual" viewing mode revealed the presence of bilateral atrophy in the medial temporal lobe in some cases, mainly limited to the hippocampus.

Functional brain imaging techniques, particularly positron emission tomography with ^{18}F-fluorodeoxyglucose (^{18}F-FDG PET), can be a useful addition to brain MRI for differential diagnosis of TEA and neurodegenerative diseases or comorbidity detection. In patients with TEA, ^{18}F-FDG PET can detect radiopharmaceutical hypometabolism in the frontal and / or temporal regions of the brain, which does not correspond to the known patterns indicating an underlying neurodegenerative disease, and the potential reversibility of such imaging data in AED therapy may be an additional discriminator in the differential diagnosis of frontotemporal dementia. A case of dynamic changes in brain function during a TEA episode was described: during the amnesic period, ^{18}F-FDG hypermetabolism was observed in the left hippocampus, followed by restoration of normal metabolism after 1 month during AEP therapy [30].

Epilepsy refers to neurodegenerative diseases, the main pathogenetic mechanisms of which are neuronal hyperexcitability and glutamate excitotoxicity, associated with cognitive impairment, which accounts for the bilateral comorbidity of epilepsy and dementia. In Alzheimer's disease, altered metabolism of the amyloid precursor protein (APP) with the formation of senile plaques, as well as τ-protein hyperphosphorylation (positions p181, p231) and neurofibrillary tangle formation in the central nervous system. Key biochemical markers of Alzheimer's disease are represented by amyloids Aβ40, Aβ42, and various epitopes of phosphorylated τ-protein (ph-Tau). Changes in the quantitative parameters of blood plasma several biomarkers (β-amyloid, PrP27-30, α-synuclein, huntingtin) can increase vascular permeability, reactive astrogliosis, oxidative stress, etc., reflecting the progression of the pathological process [31].

In a recent study, a group of scientists from the Republic of Belarus examined the levels of serum β-amyloids (Aβ1-40 and Aβ1-42), phosphorylated τ-protein (ph-Tau) of patients with pharmacoresistant epilepsy and healthy controls by using enzyme-linked immunosorbent assay (ELISA). The mean values for amyloid level of Aβ1-40, Aβ1-42 from patients with epilepsy did not differ from those in control group, comparable by age and gender, and were in the range of 0–10 pg/ml for Aβ1-40 and 30–40 pg/ml for Aβ1-42. At the same time, the ph-Tau levels in epilepsy patients were almost by 3-fold higher than in volunteers (0–10 pg/ml) comprising 28.64 pg/ml. It was concluded that the presence of hyperphosphorylated τ-protein may be associated with the subsequent formation of neurofibrillary tangles in the brain and development of cognitive disorders [32, 33].

In a retrospective study by B. Cretin et al. (2023) [34], 127 patients with TEA were followed up for 13 months. All patients' cognitive status was studied in dynamics, and 71 patients' cerebrospinal fluid (CSF) was examined for the level of amyloid β-42, ph-Tau, and total τ-protein. CSF analysis revealed the presence of ph-Tau and amyloid in 27 patients (38%), including characteristic proteins for Alzheimer's disease found in 17 (24%) cases. These patients were significantly more likely ($p<0.01$) to have various neurological disorders: Parkinsonism (42.9% vs. 20%), hyposmia (34.3% vs. 9.4%), and cognitive impairment (31% vs. 10.7%). Moreover, they had lower values of the baseline score based on the Mini-Mental State Examination (MMSE) noted in 27% vs. 28.9%, moderate cognitive impairment – 69% vs. 42.6%, and more profound atrophic changes in the hippocampus. At follow-up, patients with verified CSF changes had significantly lower ($p<0.01$) level of cognitive functions according to the MMSE scale – 27.8% vs. 28.9% [34]. Thus, a biochemical study of neurodegeneration markers can help in diagnostic search and assess disease prognosis.

A key study for epilepsy verification is long-term video EEG monitoring with sleep recording. A study by K.J. Werhahn et al (2015) [29] convincingly showed that the percentage of detected interictal epileptiform discharges in patients with epilepsy depends on the duration of video EEG monitoring: when recording EEG lasted for 20 minutes, they were detected in 7.3% of patients, 30 minutes – in 9.7%, 24 hours – in 74.6%, 48 hours – in 87.9%, 72 hours – in 96.4% [29].

As an illustration, we present our own clinical observation.

CASE REPORT / КЛИНИЧЕСКИЙ СЛУЧАЙ

Patient B., born in 1957 (65 years old), on 26.10.2022, was accompanied by relatives from Cherepovets, where the patient lives, to a multidisciplinary clinic for examination in St. Petersburg . According to daughter of patient B., 3.5 years ago the patient developed memory impairment, and a few months ago – repeated episodes of long-term twilight disorders of consciousness lasting up to 8 hours, during which patient B. left the city of residence, to other villages, performed outwardly meaningful actions, and then could not remember what happened. On 20.10.2022, the patient developed an attack of impaired consciousness with disorientation in time and space lasting more than 5 days.

Ethical aspects / Этические аспекты

The study complies with the ethical standards of the Helsinki Declaration of 1975 and its 2000 revision. The study participant was informed about the aim and main objectives and signed a written informed consent to participate in the study.

History of disease / Анамнез заболевания

In 1997, the patient B. suffered a closed craniocerebral injury. Since 2005, patient B. has been noticing a periodic increase in blood pressure to 140–160/100 mm Hg taking no antihypertensive medications. In 2022, patient B. was examined for memory impairment, and the drug memantine hydrochloride (memantine) was prescribed at a dose of

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20 mg /day, which patient B. takes constantly. Siblings of patient B. have burdened heredity for Alzheimer's disease.

History of life / Анамнез жизни

Patient B. worked as a driver, and currently retired being unoccupied; lives alone.

Neuropsychological examination / Нейропсихологическое обследование

The patient was examined by a psychologist on 26.10.2022 to analyze the level and pattern of the main mental processes to clarify the diagnosis. A neuropsychological examination was performed: a test of memorizing 10 words, pictograms, a score on the MMSE scale, the Montreal Cognitive Assessment (MoCA), the memory for numbers method, the Schulte method, the Geriatric Depression Scale (GDS).

Features of contact: when conducting a psychological study, patient B. agrees to cooperate. The contact is productive and rather confidential. Patient B. doesn't refuse to complete tasks and techniques. Neat, well-groomed. At the examination accompanied by patient B. daughter. Maladapted in society (according to patient B. daughter's stories, has difficulty in updating past events, forgets dates, absent-minded, may forget how left the apartment and came to another place).

Based on the results of the tests for assessing cognitive processes, the following data were obtained: 10 words – could not complete, Schulte method – could not complete, confused numbers. Pictograms – couldn't match images to concepts. The MoCA score was 4 points out of 30, and the MMSE score – 7 points, which corresponds to severe dementia. The geriatric scale of depression was 14 points corresponding to mild depression. Invoice – direct, reverse invoice is not available. In behavior, speech shows obvious inhibition, slowness, slurred, but without obvious pronunciation disorders, slow in tempo. Writing in slow motion, the handwriting is clearly not affected. Does not learn immediate instructions. Amnesic disorientation (patient B. says to be at home in Cherepovets), an inability to assess, analyze, and comprehend the environment. Thus, a set of cognitive disorders, a decline in higher mental functions matching the level of severe dementia was revealed.

Neurological status / Неврологический статус

At the time of examination, the patient B. is disoriented in space and time, consciousness is disturbed by the type of stunning. Cranial nerves are intact. Tendon reflexes are equal, D=S, no paresis, no pathological signs. Muscle tone is normal. No sensitive impairments. Satisfactory coordination tests. Stable in the Romberg pose. Gait not disturbed. Pelvic organ functions are preserved.

Electrocardiography / Электрокардиография

Sinus rhythm on ECG, matching heart beat rate of 69 per minute. Normal location of the cardiac electrical axis. Signs of enlarged left atrium. Incomplete blockage of the right bundle of His. Ventricular extrasystole.

Brain MRI / МРТ головного мозга

According to the brain MRI data using on the Discovery MR750W device (General Electric, USA) with a magnetic field induction of 3 T: the corpus callosum is formed, of usual shape and size. The hippocampus is characterized by a hyperintensive signal in T2-weighted images, FLAIR, the size is reduced, with altered pattern, homogeneous, and the digitation of the hippocampal head is preserved. Atrophy of both mammillary bodies is detected. The lateral ventricles are moderately dilated, symmetrical (index of the anterior horns of the lateral ventricles-31, index of bodies-30), the ventricle III is dilated (diameter 8.5 mm), the ventricle IV unaltered. Furrows, subarachnoid space of the convexus of the cerebral hemispheres is expanded. The structures of the brain stem and the chiasmal-sellar region are not changed. Paranasal sinuses are pneumatized.

Conclusion: “Bilateral hippocampal sclerosis. Atrophy of mammillary bodies. Brain atrophy. Expanded external and internal CSF spaces of a substitutive type”.

Video-EEG monitoring / Видео-ЭЭГ-мониторинг

The study was performed using the Neyron-Spektr-SM brain electrical activity analyzer (Neurosoft LLC, Russia) from 19 head leads (according to the international system of applying electrodes “10–20”).

Video-EEG monitoring was performed for 12 hours, in the state of active and passive wakefulness, during functional tests (opening and closing the eyes, 3-minute hyperventilation, photostimulation), during night sleep, with final forced awakening.

The presence of diffuse changes in brain bioelectric activity of moderate magnitude was revealed (in the form of a decreased alpha rhythm index, subdominated slow-wave activity in 6–7 Hz theta rhythm . Along with this, a periodic regional deceleration in the form of theta activity up to 5–7 Hz was recorded in the frontotemporal regions bilaterally synchronously or isolated, more often on the left side. No unqualified epileptiform activity was recorded during wakefulness. Sleep is indistinctly modulated by phases and stages, and the physiological patterns of NREM sleep are indistinctly expressed. Epileptiform activity was observed in the form of acute waves and groups of acute waves in the frontotemporal-parietal regions bilaterally synchronously and isolated in the left hemisphere. No ictal events or patterns were detected.

Laboratory examination / Лабораторное обследование

According to the laboratory examination (clinical and biochemical blood tests, general urinalysis), the data obtained were within the reference ranges.

Diagnosis / Диагноз

As a result of the examination, the diagnosis was established: “Focal epilepsy with focal motor seizures with impaired awareness – episodes of TEA. Mixed-origin encephalopathy (dyscirculatory, traumatic) with mixed

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replacement hydrocephalus and brain atrophy, bilateral hippocampal sclerosis, mammillary body atrophy, and severe cognitive impairment."

Therapy / Терапия

Levetiracetam at a dose of 1000 mg/day, memantine at a dose of 20 mg/day were prescribed. Markedly improved patient's condition was noted: consciousness was restored to a clear level, control of focal epileptic seizures (TEA) was achieved, and socialization improved due to therapy. The patient B. currently lives in a medical boarding house, supervised by medical staff, participates in public life, attends a dance school, communicates with neighbors.

CONCLUSION / ЗАКЛЮЧЕНИЕ

TGA is a clinical syndrome characterized by sudden anterograde amnesia, lasting up to 24 hours without focal neurological symptoms, not prone to relapse. Under the guise of TGA, TEA often occurs – epileptic seizures with impaired awareness of various durations, including long-term. According to the new classification of epilepsy and epileptic seizures proposed by the International Antiepileptic League (2017), TEA refers to focal seizures with impaired awareness.

TEA is characterized by recurrent episodes, combination with other epilepsy manifestations, and comorbidity with neurodegenerative diseases (dementia). Neuroimaging techniques (brain MRI, PET), psychological testing, and biochemical studies of neurodegeneration markers are important to use for the differential diagnosis between TEA and TGA.

Thus, TGA and TEA are rare phenomena in clinical practice manifested as transient cognitive amnesia disorders. Despite the common clinical sings, such conditions are pathogenetically heterogeneous, differ in prognosis and require distinct therapeutic approaches.

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